Reduced servo-control of fatigued human finger extensor and flexor muscles

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1. In healthy human subjects holding the index finger semi-extended at the metacarpophalangeal joint against a moderate load, electromyographic (EMG) activity was recorded from the finger extensor and flexor muscles during different stages of muscle fatigue. The aim was to study the effect of muscle fatigue on the level of background EMG activity and on the reflex responses to torque pulses causing sudden extensor unloadings. Paired comparisons were made between the averaged EMG and finger deflection responses under two conditions: (1) at a stage of fatigue (following a sustained co-contraction) when great effort was required to maintain the finger position, and (2) under non-fatigue conditions while the subject tried to produce similar background EMG levels to those in the corresponding fatigue trials.

2. Both the unloading reflex in the extensor and the concurrent stretch reflex in the flexor were significantly less pronounced and had a longer latency in the fatigue trials. Consequently, the finger deflections had a larger amplitude and were arrested later in the fatigue trials.

3. It is concluded that – with avoidance of 'automatic gain compensation', i.e. reflex modifications attributable to differences in background EMG levels – the servo-like action of the unloading and stretch reflexes is reduced in fatigued finger extensor and flexor muscles.

The now widely accepted 'servo-assistance theory' implies that skeletomotor output in voluntary muscle contractions is reflexly supported and controlled by afferent inflow from fusimotor-biased muscle spindles (Matthews, 1981). The increasing interest in muscle fatigue over the last few decades has led to conflicting opinions as to whether the reflex-mediated component of the skeletomotor output increases or decreases as muscle fatigue develops during a sustained isometric voluntary contraction (for review see Enoka & Stuart, 1992; Stuart & Callister, 1993). The prime support for the former alternative comes from experiments on anaesthetized, decerebrate or spinalized cats where muscle fatigue is produced by tetanic stimulation of muscle nerves or ventral roots (Nelson & Hutton, 1985; Hutton, Atwater & Nelson, 1992; Ljubisavljevic, Jovanovic & Anastasijevic, 1992). By contrast, the main support for the latter alternative comes from studies on human subjects striving to maintain a constant contraction force (Bongiovanni & Hagbarth, 1990; Bongiovanni, Hagbarth & Stjernberg, 1990; Gandevia, Macefield, Burke & McKenzie, 1990; Macefield, Hagbarth, Gorman, Gandevia & Burke, 1991; Macefield, Gandevia, Bigland-Ritchie, Gorman & Burke, 1993).

Using the microneurographic recording technique, Macefield *et al.* (1991) noted a progressive decline in spindle afferent discharge from the human pretibial muscles during a sustained foot dorsiflexion with constant force. This finding was considered to support earlier indirect evidence (Bongiovanni & Hagbarth, 1990; Bongiovanni *et al.* 1990) that a progressively declining support via the fusimotor reflex loop contributes to the 'muscle wisdom' phenomenon (Marsden, Meadows & Merton, 1983), i.e. the functionally appropriate reduction in motor unit firing rates which occurs in parallel with declining contractile force during a sustained maximal voluntary contraction (Bigland-Ritchie, Johansson, Lippold, Smith & Woods, 1983; Bigland-Ritchie, Dawson, Johansson & Lippold, 1986). One of the earlier studies indicated that waning spindle support may result not only from declining spindle afferent inflow to the spinal cord but also from increasing presynaptic inhibition or exhaustion of group Ia terminals (Bongiovanni *et al.* 1990). Results obtained in recordings from human motor axons in the ulnar and peroneal nerves during acute de-afferentation of the target muscles are consistent with the view that the autogenetic facilitatory influence provided by muscle afferents declines as muscle fatigue develops (Gandevia *et al.* 1990; Macefield *et al.* 1993).

A series of studies have been devoted to the question of whether phasic stretch and unloading reflexes are enhanced or reduced during muscle fatigue. The reports concerning

this question are seemingly contradictory. Some investigators have observed enhancements of perturbation-induced reflex EMG responses during muscle fatigue (Häkkinen & Komi, 1983; Windhorst, Christakos, Koehler, Hamm, Enoka & Stuart, 1986; Kirsch & Rymer, 1987), whereas others have observed reflex reductions (Corden & Lippold, 1989; Balestra, Duchateau & Hainut, 1992). It has also been reported that reflexes of this type are enhanced during the early stage of muscle fatigue when the subject – by an increase in effort – can still produce a desired force, whereas reflex reductions occur later when the force can no longer be maintained (Marsden, Merton & Morton, 1976; Darling & Hayes, 1983).

It is well known that when a human subject sustains a submaximal contraction of constant force to exhaustion, the whole muscle EMG activity increases gradually, apparently due to a progressive recruitment of motor units (Bigland-Ritchie *et al.* 1986; Macefield *et al.* 1991; Psek & Cafarelli, 1993); the decline in EMG activity starts when the desired force can no longer be maintained. It is also well known that the reflex gain varies automatically with the pre-existing level of motor discharge ('automatic gain compensation'; Matthews, 1986). Thus, when studying fatigue-induced changes in the efficacy of spindle inputs impinging on the motoneurone pool – rather than the readiness of the motoneurone pool to respond to any given type of input – it is essential that the pre-existing level of EMG activity is similar in the control runs (i.e. in the non-fatigued state) to that during the fatigue trials. In the present study on the unloading and stretch reflexes of human finger extensor and flexor muscles, attempts were made to fulfil this requirement. To compensate for the minor differences in background EMG activity that nevertheless occurred, the magnitude of the reflex responses was expressed as a percentage of the pre-stimulus level.

METHODS

The experiments, approved by the local ethics committee, were performed on three healthy male subjects (the authors), aged 40, 42 and 67 years.

Mechanical arrangements

The pair of antagonistic muscles studied were the extensor and flexor muscles of the index finger. The subject sat with his left forearm and pronated hand fixed against a support, which ended at the metacarpophalangeal joints. The index finger was attached to the crank arm of a torque motor with its spindle positioned co-axial to the metacarpophalangeal joint. The angular position of this joint was measured by a spindle-attached potentiometer coupled to a DC amplifier with a lamp which gave the subject a visual signal as long as he held his finger in a desired semi-extended position of 45 deg (± 2 deg). To compensate for the weight of the crank arm (resting torque 0·18 N m) he had to maintain an antigravity contraction in the index finger extensor, corresponding to about 30% of maximal force in the non-fatigued state.

EMG recordings

In all experiments the electromyographic (EMG) activity of the extensor indicis proprius was recorded by a pair of tungsten needle electrodes with 4 mm long uninsulated tips inserted about 1 cm apart in the muscle belly. In five of the endurance tests (see below), similar needle recordings were made from the index finger portion of the flexor digitorum superficialis; in all other experiments finger flexor EMG activity was recorded less selectively by a pair of surface electrodes (saline-soaked pads) placed on the volar side of the arm 7 and 10 cm proximal to the wrist joint. The amplified EMG signals (filtered between 0·3 and 5 kHz) were monitored on a loud speaker, displayed on an oscilloscope and analysed as described separately below for the endurance and reflex tests.

Endurance tests

To test the validity of previous reports and as a prelude to the subsequent reflex studies, a series of on-line recordings ($n = 20$) were made of the gradual changes in mean voltage EMG levels during different stages of muscle fatigue. In these recordings the EMG signals were full-wave rectified and 'integrated' by an RC circuit with a time constant of 0·1 s before being displayed on a thermo-writer (Graphtec Thermal Arraycorder Wr 7700; Graphtec, Tokyo) together with the goniometer signals. The subject merely lifted the index finger to the desired position and held it there until the finger yielded under the weight of the crank arm. The finger lifts were preceded either by rest periods of at least 15 min or by strong, sustained co-contractions of the finger flexor and extensor muscles lasting for 30–60 s.

Reflex tests

In thirty-seven experiments, the torque motor was used to deliver a brief rectangular torque pulse (0·25 N m; duration, 500 ms), strong enough to overcome the resting torque, thereby causing a sudden finger extension movement and an unloading of the index finger extensor. Each experiment consisted of ten trials during fatigue and ten trials on recovery from fatigue.

In the 'fatigue trials' the torque pulses were applied at about 5 s intervals during endurance tests performed after fatiguing co-contractions (see above and Fig. 1*B*, left). Occasionally, exhaustion was so pronounced that the subject could not maintain the finger position long enough for the whole series of torque pulses to be applied; he was then allowed to relax a few seconds before re-assuming the test position.

The 'non-fatigue trials' were performed after at least 15 min of rest. Guided by the audiomonitored and oscilloscope-displayed EMG signals the subject tried to induce background EMG levels in the finger extensor and flexor muscles similar to those encountered in the 'fatigue trials' (Fig. 1*B*, right). With intervening 5 s relaxation periods, ten short-lasting finger lifts were made, and each time a torque pulse was applied when appropriate EMG levels were reached. In both types of trials, the subject was kept unaware of the exact timing of the stimuli, and no attempts were made to 'resist' or 'let go'. The choice of the authors as subjects was motivated by the difficulties involved in teaching naive subjects to follow the instructions and to achieve appropriate background EMG levels in the non-fatigue trials.

Data analysis of reflex tests

For each type of trial, the goniometer deflections and the full-wave rectified (but not 'integrated') EMG signals were averaged and compared as described in the Results section. This was made off-line on a digital wave-form analyser (DATA 6100; Data

Precision, Danvers, MA, USA) coupled to an x–y plotter (Hewlett-Packard, 7475 A). An analog tape-recorder (Sangamo Sabre, VI; Springfield, IL, USA) was used for storage and display of the recorded events.

Pooled values from the experiments are reported as means ± S.E.M. Differences were evaluated by Wilcoxon's signed rank test with $P < 0{\cdot}05$ as the chosen level of significance.

RESULTS

Fatigue-induced changes in background EMG activity

With the muscles initially non-fatigued, the endurance period (time until finger drop began) lasted for at least 4–5 min. When an endurance test was preceded by a fatiguing, sustained co-contraction of the muscles involved (see Methods), the finger position could usually not be maintained for more than 60–90 s. A typical example of such an endurance test is shown in Fig. 1*A*. As the subject with increasing effort maintained the finger position, the level of EMG activity in the extensor muscle rose slowly until the moment when (in spite of continued extension effort) the finger started to drop. Towards the end of the endurance period a rising EMG activity was also recorded from needle electrodes inserted into the antagonistic finger flexor. Such rising finger flexor activity was also regularly seen in the surface recordings. As illustrated, the EMG activity in the load-bearing muscle rose from an initially moderate level up to a peak of similar height to that reached during a preceding brief maximal contraction. In the antagonist, the EMG activity at the end of the endurance period was about half of that during an attempted maximal contraction. For both muscles, the activity peaks were reached at a time when the finger position could no longer be maintained. As the finger then slowly sank down, the EMG activity of both the extensor and flexor declined.

The two panels in Fig. 1*B* illustrate the conditions under which the torque pulses (arrows) were applied in the two types of trials. The right panel shows an example of one of the ten finger lifts – performed under non-fatigued conditions – in which the subject by voluntary co-contraction tried to induce background EMG levels which approximately corresponded to those encountered during the fatigue trials (left panel).

Fatigue-induced changes in perturbation responses

Figure 2 shows a representative example of averaged perturbation-induced reflex responses under non-fatigued and fatigued conditions. When paying attention to the EMG events during the 100 ms interval following torque pulse application, the following differences can be observed: (1) the reduction of finger extensor EMG activity (the unloading reflex) starts later and is less pronounced in the fatigue trials (middle traces) and (2) a distinct stretch reflex response in the finger flexor is seen only in the non-fatigue trials (bottom traces). Another obvious difference is

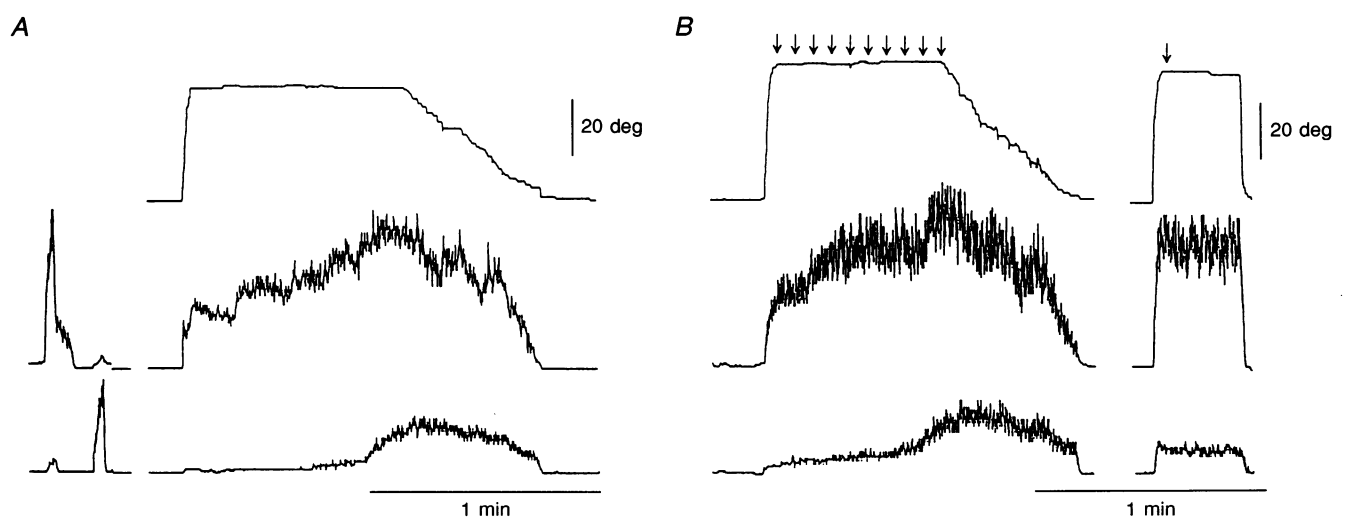

Figure 1. EMG activity during fatiguing position-holding of the index finger

Angular position of the index finger (upper traces), and mean voltage EMG activity of the extensor indicis proprius muscle (middle traces) and the antagonistic index finger flexor muscle (lower traces). *A*, EMG activity during a short-lasting attempted maximal contraction of each of the two muscles in a non-fatigued state (left panel) and during an endurance test when the finger was held semi-extended against a moderate load until the position could no longer be maintained (right panel); the endurance test was preceded by a fatiguing co-contraction (not illustrated). *B*, schematic illustration of the conditions under which the extension torque pulses were applied in the fatigue trials (left panel) and in the non-fatigue trials (right panel). The approximate timing of the torque pulses are suggested by arrows; no actual stimuli were delivered in the recordings illustrated. See text for further comments.

that the perturbation-induced finger deflections are of higher amplitude and are arrested at a later stage in the fatigue trials (upper traces).

Pooled results from the thirty-seven experiments confirmed that in the fatigue trials the perturbation-induced finger extensions were arrested later (153·1 ± 2·5 *vs.* 135·4 ± 3·3 ms, fatigue *vs.* non-fatigue; $P = 0{\cdot}0001$) and at a higher peak amplitude (18·9 ± 0·8 *vs.* 17·7 ± 1·6 deg; $P = 0{\cdot}005$) than in the non-fatigue trials (left arrows in the two panels of Fig. 2). The arrows to the right in the same figure indicate the moment when the subsequent finger flexion movement ended. In the fatigue trials, this occurred later (295·9 ± 7·4 *vs.* 251·3 ± 8·4 ms, fatigue *vs.* non-fatigue; $P = 0{\cdot}001$) and the finger came less close to the pre-stimulus position (the angular difference from this position being 12·9 ± 0·6 *vs.* 10·0 ± 0·9 deg, fatigue *vs.* non-fatigue; $P = 0{\cdot}0006$). In other words, the finger excursions were less effectively damped in the fatigue trials.

Before quantifying the averaged EMG reflex responses, the mean level of background EMG activity was calculated for a 100 ms pre-stimulus period (Fig. 2). For the extensor muscle, this level showed no significant difference ($P = 0{\cdot}16$) between the two types of trials (7·6 ± 4·9% less activity in the fatigue trials). For the flexor muscle, on the other hand, the background EMG activity was significantly ($P = 0{\cdot}0008$) higher during fatigue. Expressed in relative terms, the level was 73·6 ± 17·3% higher than in the non-fatigue trials. This difference occurred despite our attempts to mimic the fatigue levels in the non-fatigue control trials.

The latency of the extensor unloading reflex was measured from the start of the angular deflection to the point where the EMG trace started to deviate downwards from the mean pre-stimulus level. The pooled data showed that this latency was significantly longer in the fatigue trials (46·9 ± 1·8 ms *vs.* 36·6 ± 1·4 ms, fatigue *vs.* non-fatigue; $P = 0{\cdot}0001$).

To quantify the magnitude of the unloading reflex, three consecutive 20 ms time windows, starting from the onset of the reflex, were set by cursors in the non-fatigue display (four vertical lines in Fig. 2, left middle trace). For each experiment, a paired comparison was made with the corresponding fatigue trials by using identical time windows starting at the same latency (i.e. independently of the response latency in the fatigue displays). From the scattergrams of Fig. 3 it is evident that the unloading reflex within all three time windows (W1, W2 and W3) was less pronounced in the fatigue trials. For W1, the decrease in EMG activity expressed as a percentage of the pre-stimulus level was 16·1 ± 2·9% in the fatigue trials and 41·8 ± 2·3% in the non-fatigue trials ($P = 0{\cdot}0001$). The corresponding values for W2 were 40·1 ± 2·6 *vs.* 66·2 ± 3·0% ($P = 0{\cdot}0001$), and for W3, 41·8 ± 3·9 *vs.* 52·5 ± 4·8% ($P = 0{\cdot}005$). Thus, the fatigue effect was more pronounced for the early parts of the unloading reflex.

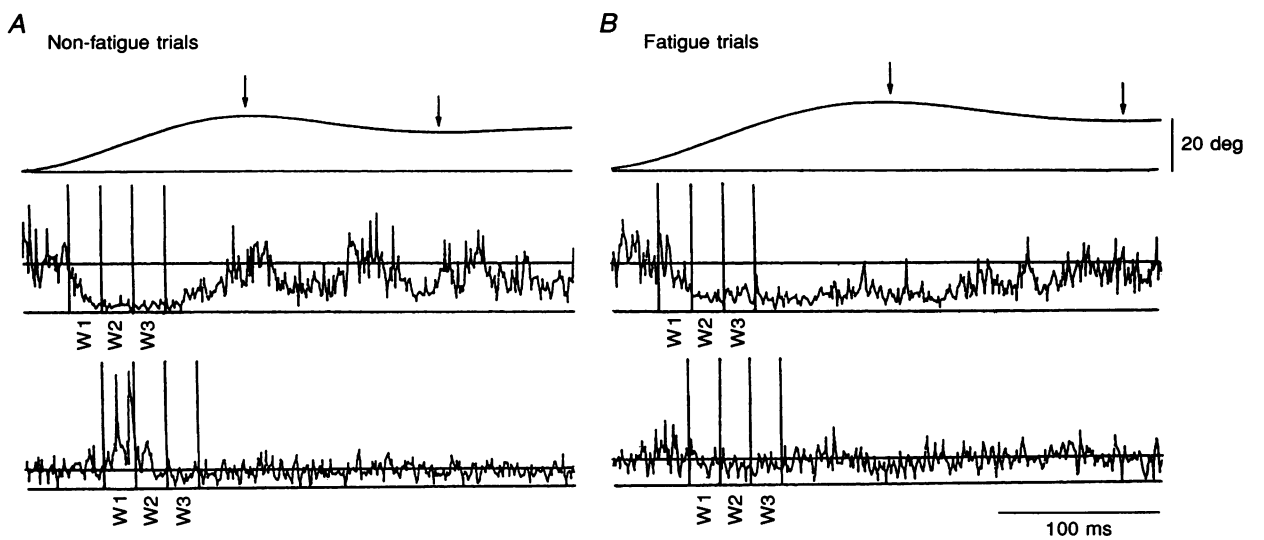

Figure 2. Effects of fatigue on perturbation-induced EMG and finger deflection responses

Comparison between perturbation-induced responses obtained in non-fatigue trials (*A*) and in the corresponding fatigue trials (*B*). The recordings show angular finger deflections (upper traces), and rectified EMG from the index finger extensor (middle traces) and the antagonistic index finger flexor (lower traces). Each trace is an average of 10 trials. The arrows point to the peak of the extension movement and to the end of the subsequent flexion movement. The horizontal lines drawn in the EMG traces indicate the zero level obtained with the muscle relaxed and the mean level of a 100 ms pre-stimulus period. The time windows (W1, W2, W3) used for quantification of the unloading and stretch reflexes are also shown (see text for further explanation). Note that both the unloading reflex in the extensor and the stretch reflex in the flexor were reduced in the fatigue trials.

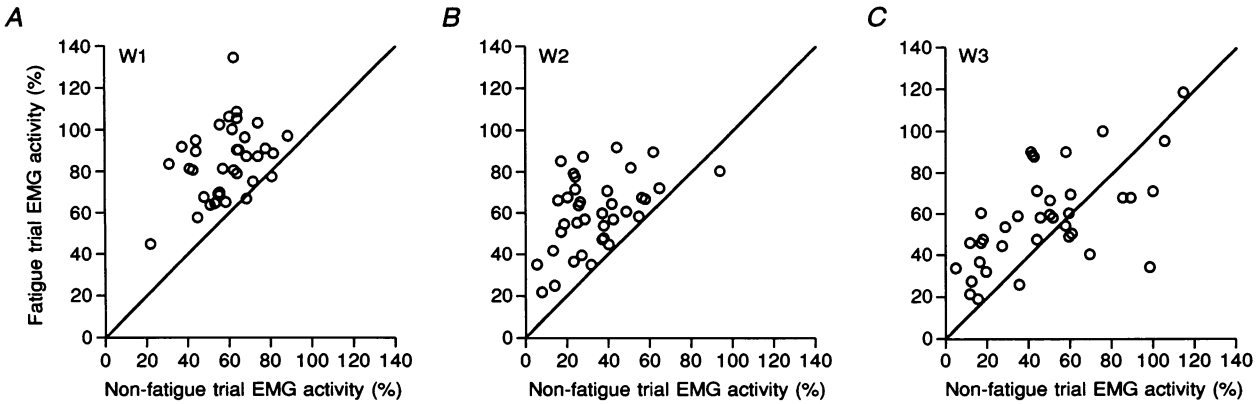

Figure 3. Effects of fatigue on the unloading reflex of the extensor indicis proprius muscle

The mean EMG activity within the three time windows shown in Fig. 2 (W1, W2 and W3) is expressed as a percentage of the pre-stimulus level. The diagonal line drawn in each scattergram indicates identical values in the two types of trials. Note that within all three time windows the EMG activity was higher – i.e. the unloading reflex was less pronounced – in the fatigue trials than in the corresponding non-fatigue trials. The number of experiments was 37.

The latency of the stretch reflex response in the finger flexor was determined by the point where the EMG trace started to deviate upwards from the mean pre-stimulus level. In fifteen of the experiments there was no recognizable stretch reflex response in the fatigue trials (example shown in Fig. 2*B*, lower trace). In five of these fifteen experiments, there was no clear-cut stretch reflex response in the non-fatigue trials either. In the remaining twenty-two experiments, the stretch reflex latency was significantly longer in the fatigue trials (57·0 ± 2·2 ms *vs.* 51·8 ± 1·5 ms, fatigue *vs.* non-fatigue; $P = 0{\cdot}0003$).

The magnitude of the stretch reflex response was quantified in a similar way as the unloading reflex, using three fixed time windows starting at the onset of the reflex response in the non-fatigue display (Fig. 2, lower traces). In this analysis, the five experiments without recognizable stretch reflex in the non-fatigue trials had to be excluded. The scattergrams of Fig. 4 show that for W1 and W2 the stretch reflex was clearly weaker in the fatigue trials, whereas for W3 there was no obvious difference. For W1, the increase in EMG activity expressed as a percentage of the pre-stimulus level was 17·7 ± 9·5% in the fatigue trials *vs.* 81·1 ± 15·7% in the non-fatigue trials ($P = 0{\cdot}0001$). The corresponding values for W2 were 6·9 ± 9·9 *vs.* 63·7 ± 14·6% ($P = 0{\cdot}0001$). For W3, there was a relative decrease of similar size in both types of trials (16·6 ± 4·6 *vs.* 19·8 ± 4·2%; $P = 0{\cdot}63$).

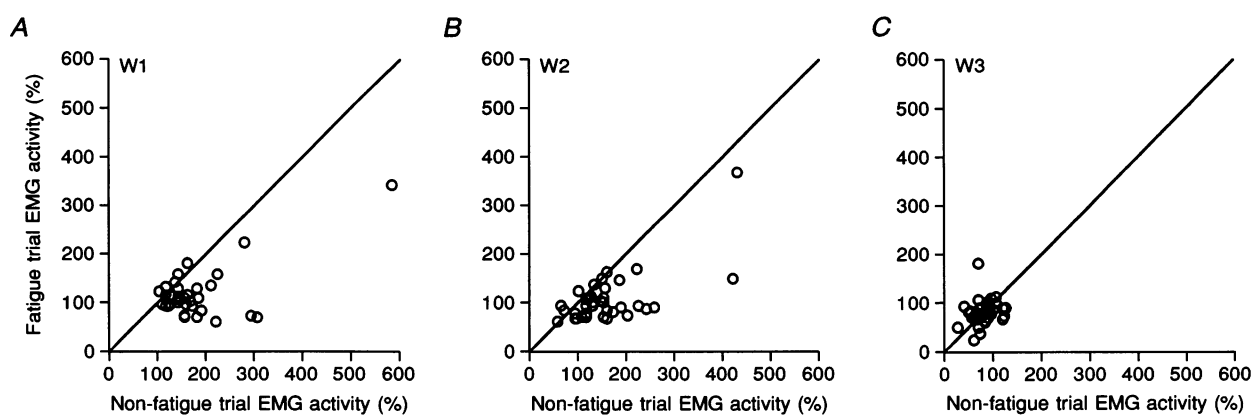

Figure 4. Effects of fatigue on the stretch reflex of the index finger flexor muscle

The mean EMG activity within the three time windows shown in Fig. 2 (W1, W2 and W3) is expressed as a percentage of the pre-stimulus level. The diagonal line drawn in each scattergram indicates identical values in the two types of trials. Note that the stretch reflex response within W1 and W2 was smaller in the fatigue trials than in the corresponding non-fatigue trials. The number of experiments was 32.

DISCUSSION

Changes in background EMG activity

The changes in EMG levels observed in the load-bearing finger extensor during the endurance tests were of the type to be expected from many earlier reports. An increasing central drive with a progressive recruitment of extensor motor units was apparently required to compensate for the fatigue-induced reduction in contractile force. This central compensation continued until the EMG had reached a similar level to that during a preceding maximal voluntary contraction. Once this critical point was reached, the phase of declining EMG activity began as the extension force needed for position holding could no longer be maintained (i.e. an expression of the 'muscle wisdom' phenomenon).

The rising finger extensor activity during the endurance tests was accompanied by an increasing EMG activity also in the finger flexor. Such activity appearing in the antagonist to a fatiguing load-bearing muscle has recently been reported by Psek & Cafarelli (1993). They propose that a central 'common drive' is responsible for this co-activation which apparently counteracts the attempted force production. In our opinion the phenomenon is most easily explained by a declining Ia reciprocal inhibition, which in turn depends on a declining Ia afferent inflow to the spinal cord during sustained voluntary contractions (Macefield *et al.* 1991).

Changes in perturbation-induced servo responses

With attempts made to avoid reflex modifications attributable to differences in pre-existing levels of EMG activity, the servo responses to load perturbations were smaller and had a longer latency in the fatigue trials. Even though cutaneous and other types of afferents may contribute to the reflex compensations for externally evoked perturbations, the primary endings of the muscle spindles are presumably the main source of such responses, at least of their earliest components. Thus, the results suggest that – with similar levels of motoneurone excitability – muscle fatigue is accompanied by a reduced efficacy in the dynamic spindle control of the skeletomotor output. With a fatigue-induced decline in the tonic autogenetic spindle support (Macefield *et al.* 1991) the disfacilitation caused by unloading will be diminished, thus explaining the reduction of the finger extensor unloading reflex. The present results provide no clue as to whether the later components of the unloading reflex (within W2 and W3) should be attributed to long-loop, possibly supraspinal reflex arcs (cf. Marsden *et al.* 1976). However, the earliest component (within W1) can safely be regarded as a spinal reflex response.

Not only the extensor unloading reflexes but also the concurrent flexor stretch reflexes were weaker and had a longer latency in the fatigue trials. It is of interest to note that the fatigue-induced stretch reflex decline occurred in spite of the fact that the pre-existing finger flexor EMG activity was stronger in the fatigue trials. Thus, since the reflex change can hardly be explained in terms of 'automatic gain compensation', it presumably depends on events occurring in the servo loop at a pre-motoneuronal level, possibly in the Ia terminals (cf. Bongiovanni *et al.* 1990).

There is evidence indicating that the rise in gross EMG activity during muscle fatigue is accompanied by a reduced firing rate of low-threshold motor units and by a recruitment of high-threshold units (for references see Enoka & Stuart, 1992). By contrast, when gross EMG activity rises during voluntary increase of contraction force the recruitment of high-threshold units is normally accompanied by an overall increase in motor unit firing rates. Since high-threshold units are less susceptible than low-threshold units to variations in spindle afferent input (for references see Burke, 1973), it cannot be ruled out that the above-mentioned difference in motor patterning to some extent can contribute to the observed reflex reductions accompanying muscle fatigue.

In both types of trials, the finger flexor stretch reflex responses had a longer latency than the extensor unloading responses (mean difference, ~10–15 ms). Possibly, this difference can be accounted for by the principle of 'automatic gain compensation', i.e. it might depend on the fact that in all reflex tests the background EMG activity was stronger in the extensor than in the flexor. At any rate, the latency difference provides no substantial evidence that the W1 component of the stretch reflex was a 'long-loop' response.

Obviously, the summed mechanical effect of the extensor unloading and the flexor stretch reflexes was to damp the perturbation-induced finger deflections. As expected from the fatigue-induced reductions in the reflex EMG responses, the finger deflections were of larger amplitude and arrested at a later stage in the fatigue trials. The results do not rule out the possibility that muscle fatigue may also induce changes in non-reflex muscle stiffness.

An objection that might be raised against the present study is that the fatigue trials were all performed at a relatively pronounced stage of muscle fatigue. However, the experiments reported were preceded by a pilot study performed during milder degrees of fatigue, i.e. before any appreciable co-contraction occurred in the antagonistic flexor muscles. In these tests, also, there was a clear tendency for perturbation-induced EMG reflex responses to be smaller during fatigued than during non-fatigued conditions, providing the background extensor EMG levels were similar in the two types of trials. No quantitative comparisons were made between the reflex reductions encountered during various stages of muscle fatigue.

It is of interest to compare the present results with those obtained in earlier studies where no attempts were made to avoid changes in reflex gain related to variations in pre-existing EMG levels. As pointed out by Matthews (1986), 'automatic gain compensation' probably arises 'just because when more motor units are active, more are available to be influenced by any given afferent input'. Thus, the organization of the motoneurone pool can fully explain the gain enhancement of stretch and unloading reflexes previously observed by several investigators during the stage of muscle fatigue when the EMG activity is rising (Marsden *et al.* 1976; Darling & Hayes, 1983; Windhorst *et al.* 1986; Kirsch & Rymer, 1987). This type of gain enhancement may well occur even though opposing, gain-reducing mechanisms simultaneously operate at the pre-motoneuronal level of the myotatic reflex arc. It is noteworthy that most previous observations on reduced servo responses in fatigued muscles were made during stages of exhaustion when the EMG activity was declining and motoneurone-based gain compensation could no longer counteract pre-motoneuronal gain-reducing events (Marsden *et al.* 1976; Darling & Hayes, 1983; Corden & Lippold, 1989; Balestra *et al.* 1992).

Implications

A pertinent question is how a fatigue-induced reflex reduction, as observed in the present study, fits with current theories on the genesis of fatigue tremor. Such tremor was not a prominent feature in the present experiments as long as the finger was loaded by the crank arm, but it became apparent when the subject with fatigued muscles made slow voluntary movements of the unloaded finger. It is now commonly recognized that the stretch reflex is a driving force in fatigue tremor (Young, 1992), and a reflex reduction would therefore be expected to diminish rather than to enhance the tremor. However, oscillations in servo loops are critically dependent on the timing of the feedback events. Possibly, the increase in latency of the stretch and unloading reflexes observed in our fatigue trials can lead to a timing of servo-loop events which under certain external load conditions provoke mechanical oscillations.

The 'overshoot' resulting from muscle fatigue resembles the type of 'overshoot' commonly exhibited by patients with cerebellar hypotonia. In such patients unloading reflexes are diminished, presumably as a consequence of reduced servo-assistance via the 'gamma loop' (Struppler, Burg & Erbel, 1973).

Reductions in servo-control of fatigued muscles may have practical consequencies of relevance for occupational medicine. The 'overshoot' resulting from sudden unloading of fatigued muscles may for example involve obvious risks when handling tools like chain-saws or other objects liable to cause unexpected perturbations. Such impairment of motor control is likely to be most apparent during pronounced fatigue, when reflex enhancements due to automatic gain compensation can no longer override presynaptic gain-reducing mechanisms.

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